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THE RÔLE OF THE TYPHOID BACILLUS IN THE PULMONARY COMPLICATIONS OF TYPHOID FEVER.*

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THE fact that typhoid bacilli are not infrequently found in the lungs during an attack of typhoid fever is well established, but the effect of their presence is still uncertain. Artaud in 1885 was the first to mention the possibility of pulmonary invasion by typhoid bacilli, and he described typhoid-like bacilli in the lungs of two typhoidal patients dying of pulmonary apoplexy. According to Curschmann, lungs, the seat of gangrene, hypostatic splenization, or even lobar pneumonia due to the pneumococcus, undoubtedly may be invaded by typhoid bacilli. When one considers the constancy with which the typhoid bacillus enters the general circulation, and also that the organism has been obtained by a number of observers (Polguère, Montier, Roque and Bancel) from lungs in a state of congestion only then it is clear that the organism quite regularly enters the lungs during typhoid fever. Roque and Bancel obtained the bacillus six times in 16 attempts by lung puncture during life from cases showing only bronchitis or pulmonary congestion. They think that the typhoid bacillus is present in greater numbers in the pulmonary than in the systemic circulation and plays an important rôle in pulmonary complications of typhoid fever.

Although the organism has been recovered repeatedly from the lung, there are in the literature but few cases in which the typhoid bacillus seems surely to have produced a definite pulmonary lesion.

Several observations have been made recently at the Pennsylvania Hospital which throw some light upon the significance of the typhoid bacillus in pulmonary complications of typhoid fever.

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Case 1.—N. B., male, white, age 22, laborer, entered the Pennsylvania Hospital (No. 1594) August 19, 1902, service of Dr. Stengel. Patient is an Italian, speaking no English, so no history can be obtained. Temperature 103.2°, pulse 80, respiration 28. The patient is a well built, well-nourished man; tongue coated; heart sounds are clear and distinct; lungs are everywhere resonant and the breath sounds are clear; liver not palpable; spleen is palpable. Abdomen is round and well formed. No tenderness is elicited. There are a few isolated rose colored spots over the abdomen. Leucocytes 1800. Typhoid bacilli agglutinated.

Patient remained in fairly good condition until September 16, when, after having had a slight cough and a slightly blood-streaked sputum for several days, he developed excessive cough, dyspnea, and hemorrhagic expectoration. The sputum is now almost pure blood and every effort to move produces a severe coughing spell. Such examination as can be made discloses subcrepitant and occasional piping rales on the right side. There is some impairment of resonance at the right base posteriorly. Few rales over left side. Heart sounds clear. Temperature rose to 104.6°, dropped in six hours to 98°, then rose on the next day to 105°.

September 17. Loud, high-pitched, squeak, systolic in time and without diastolic element is heard in the region of the apex of the heart. A few pleural friction sounds can also be heard. Temperature irregular, ranging from 105° to 97°.

September 23. Patient had a chill lasting several hours, after which he became steadily worse and died on September 24.

Autopsy (No. 299) by Dr. Longcope.

Anatomical Diagnosis.—Typhoid fever, healing ulcers of the ileum and cecum; thrombosis of branch of right pulmonary artery; abscess of right lung; acute fibrinous pleurisy; acute bronchopneumonia; acute splenic tumor; swelling of mesenteric lymph glands, parenchymatous degeneration of liver and kidneys, and infarct of right kidney.

An abstract from the autopsy notes is as follows: "The right lung is found to be bound to the pleura by fresh fibrinous adhesions, and in separating it from the parietal pleura a cavity is opened from which there escapes gas and a large quantity of dirty yellowish brown pus. The right lung is large and its lobes are bound together by old fibrous adhesions and by a fresh fibrinous exudate. The pleura over the anterior portion of the upper lobe and margin of the middle lobe is smooth and glistening, but posterior to this the surface of the lung is covered with a fine fibrous exudate, sprinkled here and there with small hemorrhages. The entire posterior portion of the lower lobe is found to be converted into an abscess cavity which measures nine cm. in length and five cm. in breadth. This cavity has been opened in removing the lung from the body. The inner walls of the abscess are covered with a grayish green necrotic material and along the walls can be seen irregular projections and villus-like processes. The walls are here and there ribbed with cords of tissue similar to the projections. Covering the lung about the cavity there is a heavy creamy fibrinous exudate which is sometimes two cm. in thickness.

"On sectioning the anterior portion of the lung, numerous reddish gray ill-defined areas of bronchopneumonic consolidation are found. In the posterior portion of the lung the cut surface is smooth reddish gray and soggy and the tissue completely consolidated. The main artery leading to the lower lobe is plugged by a firm red and gray thrombus. The large bronchi can be followed directly into the abscess cavity, where they sometimes open and are sometimes covered with fibrin and pus. The thrombus can be followed down many branches of the artery to the abscess cavity, and the ends of the vessels are found to form the villous processes described in the wall of the abscess. The bronchi at the root are filled with blood and mucus.

"The left lung is voluminous. Most of the surface is covered by old fibrous adhesions. The entire lung is semi-solid and feels as if filled with firm irregular nodules. The cut surface shows dark red hemorrhagic consolidations which stand up above the surface and present a striking contrast to the pale crepitant intervening lung tissue. The consolidations are numerous, large and confluent posteriorly, but anteriorly they are more sparsely scattered and smaller. Frequently the hemorrhagic nodules have a yellow center. The vessels are free from thrombi. The bronchi are injected and contain mucus.

"In the ileum about 80 cm. from the ileocecal valve a large, slightly swollen Peyer's patch is found, showing a rounded ulceration 0.5 cm. in diameter at its lower end. Nearing the cecum other ulcers are found, most of which are small and have clean bases. Some are partially covered with irregular extensions of the mucosa over the surface. The margins of some of the larger ulcers are deeply injected and irregular, but usually they are smooth and rounded. A few small ulcers are found in the cecum, but otherwise the large intestine shows no change.

"The spleen is much enlarged and soft. The mesenteric glands, many of which are the size of almonds, are swollen and injected.

"Iliac vessels free from thrombi."

The condition of the other organs is unimportant.

Microscopic examination.—(Zenker's fluid, paraffin, hematoxylin and eosin).

In all sections of the lungs an extensive bronchopneumonia is found principally of a desquamative type. Many alveoli are filled with desquamated epithelial cells, only a few of which are phagocytes. Elsewhere coagulated serum fills the alveoli either alone or together with desquamated epithelial cells, and a few polymorphonuclear leucocytes and red blood corpuscles. In certain foci, especially about the bronchioles, a characteristic exudate of fibrin and leucocytes extends in the alveoli, and sometimes in these areas there are hemorrhages covering the space of several alveoli and destroying the alveolar walls. The center of such foci are usually formed by a bronchus filled with fibrin, leucocytes, and nuclear fragments. The epithelium of the bronchi is desquamated and often the exudate is attached to the wall. The areas of bronchopneumonia appear to surround and extend from the bronchioles.

Sections stained by the Gram-Weigert method, fail to show any bacteria. Sections stained with Loeffler's methylene blue (Flexner's method for typhoid bacilli in tissues) show collections of well stained rod-like bodies, which in places, especially where polymorphonuclear leucocytes are most numerous, are formed into rather loose, well defined masses.

The wall of the abscess is composed entirely of a mass of necrotic material, leucocytes, fibrin, and nuclear fragments. Directly beneath this exudate, the blood vessels are seen to be much engorged, and masses of red blood cells, closely packed and scattered through the tissue, are present. The blood and deeper portion of the exudate show beginning organization. The lung beneath shows extensive desquamation of epithelial cells with here and there areas of exudative pneumonia.

A section stained by Loeffler's methylene blue shows large numbers of typical small rod-shaped bodies, especially in the tissue where necrosis has only partially taken place, in the area showing the hemorrhage. These bodies have exactly the morphology of the typhoid bacillus.

Mesenteric lymph glands show extensive endothelial proliferation.

Intestines show typical typhoid ulcerations.

Liver shows focal necrosis and fatty degeneration; the kidneys parenchymatous degeneration; and the spleen endothelial proliferation.

Bacteriologic examination.—From the abscess of the lung, pleural fluid, left lung, right lung, thrombus in lung, and kidney, B. typhosus was isolated. It was a small, rather slender bacillus, with rounded ends, rather irregular in length, often in threads, which stain fairly well in proportions of 1–2, 1–3, 1–4, 1–6. It was actively motile, did not stain by Gram, gave a positive agglutination with known typhoid serum at 1 to 100 dilution in one hour and grew characteristically in all respects like B. typhosus. Streptococcus pyogenes was isolated from the renal infarct and liver and B. lactis aërogenes from the pulmonary thrombus, kidney, liver, and renal infarction.

Summary.—After being in the hospital with a typical attack of typhoid fever 27 days, the patient suddenly develops the symptoms and signs of lung infarction, after which he passes into a marked toxemic condition and dies eight days later.

At autopsy the main artery leading to the lower lobe of the right lung is found thrombosed, and that lobe is converted almost entirely into an abscess cavity. A bronchopneumonia is found in other portions of the right lung, as well as in the left lung. A pure culture of typhoid bacillus is found in the lung abscess and in both lungs, and bacillus-like bodies alone are seen in sections stained by methylene blue but no bodies resembling organisms are seen in sections stained by the Gram-Weigert method. In the other organs the lesions are typical of typhoid fever.

As no other causative agent is to be found we feel justified in considering the case one of lung abscess due to B. typhosus, consecutive to pulmonary thrombosis.

A somewhat similar case is reported by Flexner and Harris, in which at autopsy were found typhoid fever without intestinal lesions, typhoid bacteriemia, thrombosis of the main branch of the pulmonary artery supplying the lower lobe of the right lung, gangrene of the lung, perforation of the pleura and pyopneumothorax. The typhoid bacillus was recovered from the consolidated right lung, the spleen, the liver, and the left lung. No other cases of lung abscess or gangrene in typhoid fever with bacteriological reports have been found in the literature.

From the foregoing cases we feel justified in concluding that B. typhosus is capable of causing pulmonary abscess and gangrene in lung tissue, already the seat of hemorrhagic infarction. That the organism may invade an infarcted area of the lung without causing abscess formation is shown by another case where the pulmonary artery was occluded by a thrombus, typhoid bacilli alone being isolated from the infarcted area. In this case there was no clinical evidence of the infarction. Sections through the pulmonary artery at the site of thrombosis stained with methylene blue showed in several places rod-like bodies lying between the thrombus and the wall of the pulmonary artery. No such bodies could be found in the lungs.

Bronchopneumonia is found quite often in the lungs of patients dying of typhoid fever. When the various inflammatory lesions produced by the typhoid bacillus are considered it would seem reasonable to suppose that this organism can produce bronchopneumonia. There are, however, but few observations to support this idea.

Chantemesse and Widal, who were the first to investigate the subject, obtained the organism from the lung in two cases and concluded that certain forms of bronchopneumonia, found in typhoid fever, are to be considered as specific manifestations of the disease. Finkler expressed the opinion that B. typhosus causes splenization and bronchopneumonic solidification of similar appearance, without peribronchial solidification and without a tendency to suppuration. Bruneau considers that bronchopneumonia may be caused by a localization of typhoid bacilli in the bronchi or lungs. Lepine and Lyonnett found acute bronchopneumonia in the lungs of a dog killed 10 days after injection into the trachea of attenuated cultures of B. typhosus.

Several investigators have isolated the bacillus from bronchopneumonic lungs. Gradwohl found it in pure culture in one case. Bancel, who has made an extensive study of the question of the presence of typhoid bacilli in the lung, concludes that in bronchopneumonia other organisms are usually associated. He isolated by lung puncture B. typhosus and a coccus from a bronchopneumonic lung of a typhoid fever patient.

In six autopsies upon typhoid fever cases at the Pennsylvania Hospital in which cultures were taken from bronchopneumonic areas, the typhoid bacillus was found only once, namely, in the case (Case No. 1) reported here, in which it was obtained in pure culture. As no other organism could be found in a careful microscopic search of the tissues, we consider the bronchopneumonia as caused by the typhoid bacillus. In the other five cases the common pyogenic cocci were isolated.

The rôle played by the typhoid bacillus in lobar pneumonia complicating typhoid fever has been much discussed. The earlier writers, Karlinski, Bruneau, and others, considered that lobar pneumonia associated with typhoid fever was always due to a secondary invasion of the pneumococcus. All investigators have found the pneumococcus frequently present in such conditions. It was found three times in the six cases of lobar pneumonia occurring in typhoid fever patients that have recently come to autopsy at the Pennsylvania Hospital. B. typhosus was isolated in but one case, in which both organisms were obtained. Busquet showed that the typhoid bacillus and pneumococcus can exist together in the circulating blood of patients suffering from pneumonia as a complication of typhoid fever. He obtained both organisms simultaneously by blood culture in two cases.

A similar case has been studied at the Pennsylvania Hospital. A patient entered the hospital on the 10th day of a severe typhoid fever. Three days later his temperature became subnormal and remained so until his death, seven days after his admittance to the ward. No definite signs of pneumonia were noted. On the 16th day of his disease 10 c.c. of blood were drawn from his arm vein and distributed among six flasks containing 100 c.c. of broth each, and two flasks containing 100 c.c. of litmus milk each.

Smears made from flasks after 24 hours at 37°C. showed bacilli, and in all but two flasks, one of milk and one of broth, cocci were seen. By transplanting on human blood agar the organisms were found to be typical typhoid bacilli, giving a positive serum reaction up to 1 to 200 dilution in one hour with known typhoid serum, and pneumococci.

In another case of typhoid fever complicated by lobar pneumonia, B. typhosus was obtained in pure culture from the circulating blood on the 17th day of disease, while at autopsy, four days later the pneumococcus alone was obtained from the heart's blood. Here no doubt the organisms were coexistent in the general circulation during life.

Although there is no doubt that the pneumococcus is the usual cause of lobar pneumonitis in typhoid fever, there is considerable evidence that such a lesion may be caused by the typhoid bacillus. Curschmann expressed the opinion that the typhoid bacillus may cause a true lobar pneumonitis, although this must occur very rarely. One of the earliest workers on this subject, Polguère, expressed a similar belief, while other investigators (Karlinski, Fraenkel, Bruneau, Montier, and others) consider a true typhopneumonia as unproved or even impossible.

Mallory describes a case of fibrinous pneumonia occurring during typhoid fever in which many typhoid-bacillus-like bodies and a few pneumococci were found in sections. From his study of the case he considers that the presence of the typhoid toxin may influence by production of epithelioid cells the character of pneumonia produced by the pneumococcus.

A case of pneumonia, lobar in type, recently came to autopsy at the Pennsylvania Hospital; from the lung was cultivated a pure culture of the B. type of the paratyphoid bacillus. We believe from several points of evidence that this organism was the cause of the pneumonia.

Case 2.—J. N., Italian laborer, age 21 years, entered the service of Dr. Lewis at the Pennsylvania Hospital, April 25, 1904 (No. 298). His illness began five days before with fever, chills, and pain in right side. At time of entrance the patient expectorated bloody sputum. Physical examination showed typical signs of lobar pneumonia. The leucocytes were however only 5,720. Temperature was somewhat irregular, ranging between 100° and 105°. He died on the eighth day of his disease.

At the autopsy there was found a lobar pneumonitis; acute fibrinous and chronic fibrous pleurisy; acute splenic tumor; cloudy swelling and fatty degeneration of liver; hemorrhages into the gastric mucosa.

The left lung showed no pneumonia, but the right presented an extensive lobar consolidation, crepitus being obtained only along the posterior border· An abstract of the autopsy notes is as follows: Beneath the right pleura small distinct hemorrhages are seen as dark red spots. The cut surface of the right lung is somewhat uneven and has a dark purple color, through which small bronchioles can be seen. From these bronchioles little masses of yellow purulent material can be expressed. Pinkish gray streaks of connective tissue are seen running through the cut surface, which appears much like the cut surface of a congested spleen. The lung presents a hemorrhagic appearance.

Sections of the consolidated part of the right lung, (Zenker's fluid, paraffin, hematoxylin and eosin) present the following picture: The alveolar arrangement of the lung can be made out. Throughout the section there are scattered diffusely many polymorphonuclear leucocytes. These, together with fibrin, coagulated material, a few small round cells, and a few desquamated epithelial cells, usually fill the alveoli. The alveolar capillaries are engorged with blood and stand out prominently. In and directly under the pleura, are large collections of red blood corpuscles. In two or three areas masses of red blood cells fill several adjoining alveoli. In other areas many red blood cells are scattered diffusely through the alveoli. In a few places masses of blood appear to replace the lung tissue. The blood vessels are everywhere filled, and in several of them the blood has a homogeneous conglomerate appearance suggestive of red blood corpuscle thrombi. There is an amount of yellow and black pigment present.

Sections through the same part of the lung as before described, stained with methylene blue according to the method advised by Flexner for staining, typhoid bacilli in the tissues, give the following picture: In a part of the section that contains the largest amount of free blood, there are seen blue staining bacillus-like bodies in considerable numbers. These bodies have the morphology of the typhoid bacillus, and are in places arranged in fairly dense masses. Similar sections stained by Gram stain show nothing resembling bacilli, nor is there anything appearing like the pneumococcus to be seen. The fibrin is well stained but in no place does it resemble the bacillus-like bodies described above, being coarser and in longer strands.

Cultures were made on agar plates at time of autopsy from the heart's blood and consolidated area of the right lung, and a tube of human blood agar was inoculated from the right lung. The plate inoculated from the heart's blood and the blood agar tube remained sterile after incubation for several days at 37°C. The plate inoculated from the consolidated lung after 24 hours incubation at 37°C. showed three rather dense, round, surface colonies one to three mm. in diameter, which are distinctly raised and have a dense center and thin edges, where the colonies are semitranslucent. There are about 15 deep colonies. Coverslips from the surface colonies show somewhat irregular, rather small bacilli. The organism obtained from the lung in pure culture proved to be B. paratyphosus, type B. It was very motile, fermented mannite readily, but did not ferment lactose or saccharose. It was negative to Gram's stain. Litmus milk was quite blue after four days incubation at 37° C. The organism gave a positive agglutination reaction with serum from a known case of paratyphoid infection, agglutinating in 1 to 100 dilution in one hour.

In coverslips from the lung there was seen a few rather long narrow bacilli, and a small number of cocci in pairs resembling the pneumococcus.

Summary.—Here is a man dying on the eighth day of a disease in which typical physical signs of pneumonia were found. He had bloody expectoration, and a low leucocyte count. At autopsy there is found a hemorrhagic type of lobar pneumonitis. From the lung tissue a pure culture of the B. type of the paratyphoid bacillus is obtained. No other organisms can be found in the tissues, although smears from the lung show pneumococcus-like organisms.

If this case is, as it seems to be, one of lobar pneumonitis, caused by the paratyphoid bacillus, it would encourage the belief that such a lesion may be produced by another organism of the same group and one so much resembling it in its biology and pathogenicity as B. typhosus

There are in the literature a number of cases in which the presence of the typhoid bacillus in consolidated lung tissue was proved, while the search did not reveal the pneumococcus. Foà and Bordoni-Uffreduzzi were the first to report a case in which a pure culture of typhoid bacilli was found in inflammatory pulmonary consolidation of lobar extent. They considered it evidence that such a lesion can be produced by other causes than the micrococcus lanceolatus. Anton and Fütterer isolated the bacillus and an undetermined coccus from the lung of a case in which pneumonia developed during typhoid fever. Bruhl reports a case which showed the signs of typhoid fever only after eight days of what seemed a typical attack of pneumonia. The patient was known to have been previously exposed to typhoid infection. B. typhosus was isolated from the lung, and no pneumococci could be found, even by inoculation of mice. The blood of the patient remained sterile. The author considers the case one of a true typhoid bacillus pneumonia. He also quotes a case of Widal in which B. typhosus was isolated by lung puncture from a case resembling pulmonary tuberculosis. Castaigne reports a similar case, in which lung puncture made two days after the onset of pneumonia showed a pure culture of typhoid bacilli. Inoculation of mice here also failed to show any pneumococci. Finally, Glacer reported a case of frank pneumonia from which a pure culture of typhoid bacilli was obtained by lung puncture and in the lung tissues of which only bacilli could be found.

The experimental work upon this subject has been too small to be of much service. Lepine and Lyonnet injected attenuated cultures of typhoid bacilli into the tracheas of eight dogs. Three died very soon after the injection, and enormous hyperemia with hemorrhages were found. One dog was killed 10 days after the injection and the lungs showed an acute bronchopneumonia. Four dogs were killed 15, 17, 24 and 30 days after inoculation, and all showed more or less thickening of the alveolar walls. No true lobar pneumonia was found.

The foregoing cases seem to us to be sufficient to warrant the belief that both the typhoid bacillus and the paratyphoid bacillus, type B, are capable of producing pulmonary inflammation lobar in extent.

The hemorrhagic condition of the pulmonary lesion in our case seems especially worthy of emphasis. The appearance of the lung at time of autopsy was obviously different from that of an ordinary lobar pneumonia. Polguère noticed the hemorrhagic condition of pulmonary lesions of typhoid fever and said that bronchopneumonia in typhoid fever could be produced either by the pneumococcus or by the typhoid bacillus; that caused by the latter being the more hemorrhagic. No other investigator of the subject of typhopneumonia has since mentioned this point.

There is considerable evidence that the pulmonary lesions of typhoid fever are especially hemorrhagic, however, from the condition of the sputum in almost all of the cases reported. Anton and Fütterer appear to be the first to note that the sputum is hemorrhagic in certain cases of pneumonia occurring during typhoid fever. These authors state that the patient's mouth was full of blood the day after the onset of pneumonia in the sixth day of typhoid fever. Sahli reports a case of typhoid fever which he considers complicated by pneumonia rather than lung infarction, in which the sputum was first bloody and then serous. The typhoid bacillus was isolated from the sputum. The sputum is described as hemorrhagic in such cases by Fraenkel, Diendonné, Edel and Glaser. Stühlern, who isolated the typhoid bacillus twice from the sputum of cases with signs of pulmonary consolidation, describes it in both cases as decidedly hemorrhagic. He considers the bacillus a secondary invader of the lung, and questions whether the hemorrhagic condition of the sputum may not be an effect of the presence of the typhoid bacillus in the lung.

Lepine and Lyonnett, in their experimental work, noted the production of enormous hyperemia with hemorrhages in the lungs following the intratracheal injections of attenuated typhoid bacilli.

The fact that the typhoid bacillus may be present in the lungs in the pulmonary complications of typhoid fever, makes it probable that it is not infrequently present also in the sputum under such circumstances. It has been obtained by a number of observers from this source. In 1894 Sahli reported that he obtained the bacillus in pure culture from the sputum. Edel examined the sputum of 11 cases of typhoid fever, 10 of which had bronchitis and one had pneumonia. From the case of pneumonia the typhoid bacillus was obtained. The most extensive study of this question has been made by Jehle. He examined the sputum of 15 cases of typhoid fever and found the bacillus in the sputum nine times, twice in pure culture. He also examined the bronchial secretion at autopsy, and isolated the organism 12 times. This includes five cases of hemorrhagic infiltration of the lung all of which showed the presence of the bacilli in the bronchial secretion. The author concludes that the typhoid bacillus is present regularly in the sputum and bronchial secretions in cases of typhoid fever complicated by pneumonic or bronchopneumonic processes, and that it exists there less regularly in the sputum of cases with simple bronchitis only. Diendonné, Glacer and recently Rau have reported single cases of pneumonia complicating typhoid fever and showing the organism in the sputum. None were in pure culture.

The organism was recently isolated from the sputum of a case of typhoid fever in the Pennsylvania Hospital.

Case 3.—P. B., Italian, age 25, entered Dr. Scott's service on February 26, 1905, on the 10th day of his disease. He complained of headache, malaise and abdominal pain. Rose spots were seen, his spleen was not palpable, and the temperature was 101°. Typhoid bacilli agglutinated by the patient's serum; leucocytes 5,200. On March 10, the 23d day of disease, pain above the liver was complained of. A few fine crepitant rales were heard on inspiration on the right side between the anterior and posterior axillary lines. On the 28th day of disease there was a well marked pneumonia over the entire upper part of the right lobe. Leucocytes 11,100. The next day blood stained sputum was expectorated. On March 18 a blood culture was taken. On the same day cultures of the sputum were made, a technique similar to that of Edel being followed. A fairly large mass of thick tenacious sputum, blood-tinged and deeply rusty throughout, raised by coughing, was collected in a sterile Petri dish and taken directly to the laboratory. The mouth was not previously washed out. In the laboratory the sputum was washed in five successive dishes of sterile salt solution and inoculated on plates of plain agar in two series of three plates each. Plates were also made on glucose agar, in the same dilution as the second in the series. All plates were much overgrown by bacteria except one glucose agar plate, and the third plates of the agar series. From these plates all colonies resembling the typhoid bacillus were transplanted. Only two, one from a glucose agar plate, and one from a plain agar plate, proved to be B. typhosus. The organism is agglutinated by the serum of a known case of typhoid fever in 1 to 200 dilution in one hour. From the blood an organism similar in every respect was obtained in pure culture.

The occurrence of the typhoid bacillus in the sputum shows the importance of taking proper means to prevent infection through this source.

CONCLUSIONS.

The typhoid bacillus not infrequently invades the lung during typhoid fever.

It may invade areas of the lung already the seat of hemorrhagic infarction and there produce abscess formation and gangrene.

The organism may cause bronchopneumonia.

Lobar pneumonia as a complication of typhoid fever is usually due to the pneumococcus. This organism may be present as a general infection in the circulating blood simultaneously with B. typhosus.

It is probable that both B. typhosus and B. paratyphosus, type B, can produce a massive pneumonia, lobar in type. When these organisms are the causative factors, the pneumonia is of a peculiar hemorrhagic character, which may be recognized clinically from the bloody nature of the sputum.

The typhoid bacillus is not infrequently found in the sputum of typhoid fever patients with pulmonary complications. This fact should be emphasized in order that spread of the disease by this means may be prevented.

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